WEST

The Contents of Case 09464303

Qnum	Query	DB Name	Thesaurus	Operator	Plural
Q1	(Stahl)[IN] OR (collard)[IN]	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES
Q2	Q1 and complement	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES
Q3	mbl or (mannose adj binding adj lectin)	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES
Q4	Q3 and (3f8 or 2a9 or hmbl1.2 or hb-12621 or hb-12620 or hb-12619)	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES

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conditions, LCL induced titers of IFN-.ga[redacted] corresponding to >20,000 IFN-.alpha. units/mL medium, higher than [redacted]d. with other tested, established IFN-.gamma. inducers. Other desirable properties of this lectin, as discussed, also suggest that it will be of value for efficient large-scale IFN-.gamma. prodn.

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	38.79	38.94
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
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NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
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=> s Stahl G?/au or collard C?/au
L1 964 STAHL G?/AU OR COLLARD C?/AU

=> s l1 and complement
L2 168 L1 AND COMPLEMENT

=> s l2 and ((MBL) or (mannose (1N) binding (1N) lectin))
L3 41 L2 AND ((MBL) OR (MANNOSE (1N) BINDING (1N) LECTIN))

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 21 DUP REM L3 (20 DUPLICATES REMOVED)

WO | 2001 12217 A1

=> dis l4 1-21 ibib abs

L4 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:137043 CAPLUS
DOCUMENT NUMBER: 134:188227
TITLE: Inhibitors of the lectin **complement** pathway (LCP) and their use
INVENTOR(S): **Stahl, Gregory L.**; Lekowski, Robert
PATENT ASSIGNEE(S): The Brigham and Women's Hospital, Inc., USA
SOURCE: PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001012212	A1	20010222	WO 2000-US22123	20000814

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-148815P P 19990813
AB The invention relates to methods and products for regulating lectin **complement** pathway assocd. **complement** activation. The methods include both in vitro and in vivo methods for inhibiting lectin **complement** pathway assocd. **complement** activation. The methods are accomplished by contacting a mammalian cell having surface exposed **MBL** ligand with an effective amt. of a mannan binding lectin (**MBL**) receptor antagonist to inhibit lectin **complement** pathway assocd. **complement** activation. The mannan binding lectin receptor antagonist may be administered to a subject to prevent cellular injury mediated by lectin **complement** pathway assocd. **complement** activation. The products of the invention include compns. of a mannan binding lectin receptor antagonist. The mannan binding lectin receptor antagonist is an isolated mannan binding lectin that selectively binds to a human mannan binding lectin epitope and that inhibits lectin **complement** pathway assocd. **complement** activation.
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 21 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001259476 MEDLINE
DOCUMENT NUMBER: 21136395 PubMed ID: 11238665
TITLE: A keratin peptide inhibits **mannose-binding lectin.**
AUTHOR: Montalto M C; **Collard C D**; Buras J A; Reenstra W R; McClaine R; Gies D R; Rother R P; **Stahl G L**
CORPORATE SOURCE: Department of Anesthesiology, Perioperative and Pain Medicine, Center for Experimental Therapeutics and Reperfusion Injury, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: F32 HL-103870 (NHLBI)
HL-03854 (NHLBI)
HL-56086 (NHLBI)
SOURCE: JOURNAL OF IMMUNOLOGY, (2001 Mar 15) 166 (6) 4148-53.
Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010521
Last Updated on STN: 20010521
Entered Medline: 20010517
AB **Complement** plays a significant role in mediating endothelial injury following oxidative stress. We have previously demonstrated that the lectin **complement** pathway (LCP), which is initiated by deposition of the **mannose-binding lectin** (**MBL**), is largely responsible for activating **complement** on endothelial cells following periods of oxidative stress. Identifying functional inhibitors that block **MBL** binding will be useful in characterizing the role of the LCP in disease models. The human cytokeratin peptide SFGSGFGGGY has been identified as a molecular mimic of N-acetyl-D-glucosamine (GlcNAc), a known ligand of **MBL**. Thus, we hypothesized that this peptide would specifically bind to **MBL** and functionally inhibit the LCP on endothelial cells following oxidative stress. Using a BIAcore 3000 optical biosensor, competition experiments were performed to demonstrate that the peptide SFGSGFGGGY inhibits binding of purified recombinant human **MBL** to GlcNAc in a concentration-dependent manner. Solution affinity data generated by BIAcore indicate this peptide binds to **MBL** with an affinity (K(D)) of 5 x 10(-5) mol/L. Pretreatment of human serum (30%) with the GlcNAc-mimicking peptide (10-50 microg/ml) significantly attenuated **MBL** and C3 deposition on human endothelial cells subjected to oxidative stress in a dose-dependent manner, as demonstrated by cell surface ELISA and confocal microscopy. Additionally, this decapeptide sequence attenuated **complement**-dependent VCAM-1 expression following oxidative stress. These data indicate that a short peptide sequence that mimics GlcNAc can specifically bind to **MBL** and functionally inhibit the proinflammatory action of the LCP on oxidatively stressed endothelial cells.

L4 ANSWER 3 OF 21 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001481748 MEDLINE
DOCUMENT NUMBER: 21400864 PubMed ID: 11509633
TITLE: Human IgA activates the **complement** system via the mannan-binding lectin pathway.
AUTHOR: Roos A; Bouwman L H; van Gijlswijk-Janssen D J; Faber-Krol M C; **Stahl G L**; Daha M R
CORPORATE SOURCE: Department of Nephrology, Leiden University Medical Center, Leiden, The Netherlands.. A.Roos@LUMC.NL
SOURCE: JOURNAL OF IMMUNOLOGY, (2001 Sep 1) 167 (5) 2861-8.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20010830
Last Updated on STN: 20020122
Entered Medline: 20011205

AB The recently identified lectin pathway of the **complement** system, initiated by binding of mannan-binding lectin (**MBL**) to its ligands, is a key component of innate immunity. **MBL**-deficient individuals show an increased susceptibility for infections, especially of the mucosal system. We examined whether IgA, an important mediator of mucosal immunity, activates the **complement** system via the lectin pathway. Our results indicate a dose-dependent binding of **MBL** to polymeric, but not monomeric IgA coated in microtiter plates. This interaction involves the carbohydrate recognition domain of **MBL**, because it was calcium dependent and inhibited by mannose and by mAb against this domain of **MBL**. Binding of **MBL** to IgA induces **complement** activation, as demonstrated by a dose-dependent deposition of C4 and C3 upon addition of a **complement** source. The **MBL** concentrations required for IgA-induced C4 and C3 activation are well below the normal **MBL** plasma concentrations. In line with these experiments, serum from individuals having mutations in the **MBL** gene showed significantly less activation of C4 by IgA and mannan than serum from wild-type individuals. We conclude that **MBL** binding to IgA results in **complement** activation, which is proposed to lead to a synergistic action of **MBL** and IgA in antimicrobial defense. Furthermore, our results may explain glomerular **complement** deposition in IgA nephropathy.

L4 ANSWER 4 OF 21 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2001512912 MEDLINE
DOCUMENT NUMBER: 21444721 PubMed ID: 11560858
TITLE: Inhibition of **mannose-binding lectin** reduces postischemic myocardial reperfusion injury.
AUTHOR: Jordan J E; Montalto M C; **Stahl G L**
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: HL-10346 (NHLBI)
HL-10387 (NHLBI)
HL-52886 (NHLBI)
HL-56086 (NHLBI)
SOURCE: CIRCULATION, (2001 Sep 18) 104 (12) 1413-8.
Journal code: DAW; 0147763. ISSN: 1524-4539.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20010919
Last Updated on STN: 20011008
Entered Medline: 20011004

AB BACKGROUND: **Complement** consists of a complex cascade of proteins involved in innate and adaptive immunity. The cascade can be activated through 3 distinct mechanisms, designated the classical, alternative, and lectin pathways. Although **complement** is widely accepted as participating in the pathophysiology of ischemia-reperfusion injury, the specific role of the lectin pathway has not been addressed. METHODS AND RESULTS: Monoclonal antibodies (mAbs; P7E4 and 14C3.74, IgG1kappa isotypes) were raised against rat **mannose-binding lectin** (rMBL). Both mAbs recognized rMBL-A by Western analysis or surface plasmon resonance. P7E4, but not 14C3.74, exhibited a concentration-dependent inhibition of the lectin pathway, with maximal effect at 10 &mgr;g/mL. In vivo, rats were subjected to 30 minutes of left coronary artery occlusion and 4 hours of reperfusion. **Complement** C3 deposition was greatly attenuated in hearts pretreated with P7E4 compared with 14C3.74-treated hearts. Pretreatment with P7E4 (1 mg/kg) significantly reduced myocardial creatine kinase loss (48%), infarct size (39%), and neutrophil infiltration (47%) compared with 14C3.74-treated animals. In addition, P7E4 pretreatment significantly attenuated the expression of proinflammatory genes (intercellular adhesion molecule-1, vascular cell adhesion molecule-1, and interleukin-6) after ischemia-reperfusion. CONCLUSIONS: The lectin **complement** pathway is activated after myocardial ischemia-reperfusion and leads to tissue injury. Blockade of the lectin pathway with inhibitory mAbs protects the heart from ischemia-reperfusion by reducing neutrophil infiltration and attenuating proinflammatory gene expression.

L4 ANSWER 5 OF 21 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2001499911 MEDLINE
DOCUMENT NUMBER: 21433399 PubMed ID: 11549596
TITLE: Endothelial oxidative stress activates the lectin **complement** pathway: role of cytokeratin 1.
AUTHOR: **Collard C D**; Montalto M C; Reenstra W R; Buras J A; **Stahl G L**
CORPORATE SOURCE: Department of Anesthesiology, Perioperative, and Pain Medicine, Center for Experimental Therapeutics and Reperfusion Injury, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: F32-HL103870 (NHLBI)
HL-03854 (NHLBI)
HL-52886 (NHLBI)
SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (2001 Sep) 159 (3) 1045-54.
Journal code: 3RS; 0370502. ISSN: 0002-9440.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20010911
Last Updated on STN: 20011015
Entered Medline: 20011011

AB Oxidative stress increases endothelial **mannose-binding lectin** (**MBL**) binding and activates the lectin **complement** pathway (LCP). However, the molecular mechanism of **MBL** binding to the endothelium after oxidative stress is unknown. Intermediate filaments have been previously reported to activate the classical **complement** pathway in an antibody-independent manner. We investigated whether oxidative stress increases human umbilical vein endothelial cell (HUVEC) cytokeratin 1 (CK1) expression and activates the

LCP via **MBL** binding to CK1. Reoxygenation ([redacted] hours, 21% O(2)) of hypoxic HUVECs (24 hours, 1% O(2)) significantly increased CK1 mRNA (in situ hybridization) and membrane protein expression [enzyme-linked immunosorbent assay (ELISA)/confocal microscopy]. Incubating human serum (HS) with N-acetyl-D-glucosamine or anti-human **MBL** monoclonal antibody attenuated **MBL** and C3 deposition on purified CK1 (ELISA). CK1 and **MBL** were co-immunoprecipitated from hypoxic HUVECs reoxygenated in HS. Treatment with anti-human cytokeratin Fab fragments attenuated endothelial **MBL** and C3 deposition after oxidative stress (ELISA/confocal microscopy). We conclude that: 1) endothelial oxidative stress increases CK1 expression, **MBL** binding, and C3 deposition; 2) inhibition of **MBL** attenuates purified CK1-induced **complement** activation; and 3) anti-human cytokeratin Fab fragments attenuate endothelial **MBL** and C3 deposition after oxidative stress. These results suggest that **MBL** binding to endothelial cytokeratins may mediate LCP activation after oxidative stress.

L4 ANSWER 6 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:267818 BIOSIS
DOCUMENT NUMBER: PREV200100267818
TITLE: Epitope mapping monoclonal antibodies against human **mannose binding lectin**.
AUTHOR(S): Zhao, Hui (1); **Stahl, Gregory L.** (1)
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA, 02115 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A685. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **MBL** plays an important role in **complement** activation following endothelial oxidative stress. We have generated a panel of monoclonal antibodies (3F8, hMBL1.2 and 2A9) against human **MBL**. These antibodies are functional inhibitors, which can attenuate **MBL**-dependent C3 deposition after endothelial oxidative stress. The affinity of Fab fragments and whole IgG antibodies to **MBL** were very similar. However, Fab fragments of hMBL1.2 or 2A9 did not inhibit C3 deposition in a **MBL** dependent assay. Further, F(ab)2 fragments of 2A9 or hMBL1.2 were functionally much less effective compared to whole IgG, suggesting that steric hindrance of these two antibodies are important for their inhibition of **MBL** binding. Fab and F(ab)2 fragments of 3F8, on the other hand, were functionally as effective as the whole IgG. All three functional antibodies (3F8, hMBL1.2 and 2A9) bind to the carbohydrate recognition domain (CRD) of **MBL** based on protein sequencing and Western analysis of proteolytic fragments of **MBL**. **MBL** constructs consisting of sequential deletion of N- or C-terminal amino acids of the CRD region showed that the antibodies recognized different epitopes. Two disulfide bonds within the **MBL** monomer. Cys155 to Cys244 and Cys222 to Cys236 aid in stabilization of the CRD. Single, double and triple mutations of these cystidines showed that the disulfide bonds played a role in forming discontinuous epitopes for 3F8 and hMBL1.2. Epitope maps of these antibodies were further confirmed by biopanning using the FliTrx random peptide display library. Generating monoclonal antibodies against **MBL** will aid in the structure/function analysis of **MBL** and its role in inflammatory diseases.

L4 ANSWER 7 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:257627 BIOSIS
DOCUMENT NUMBER: PREV200100257627
TITLE: Regulation of pro-inflammatory genes by the lectin **complement** pathway following myocardial ischemia-reperfusion.
AUTHOR(S): Jordan, James E. (1); Montalto, Michael C. (1); Lopes da Rosa, Jessica R. (1); **Stahl, Gregory L.** (1)
CORPORATE SOURCE: (1) Brigham and Women's Hospital, 75 Francis St., Boston, MA, 02115 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A463. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Reperfusion of ischemic myocardium initiates an inflammatory-like process leading to additional tissue injury. Following myocardial ischemia and reperfusion (MI/R), **complement** is activated with a subsequent increase in pro-inflammatory cytokines and adhesion molecule gene expression. Increasing evidence suggests that the lectin **complement** pathway (LCP) is involved in initiating the **complement** cascade following MI/R. In this study, we assessed the result of inhibiting **complement** activation via the LCP on the expression of inflammatory genes (cytokines and adhesion molecules) following MI/R. Male Sprague-Dawley rats were subjected to 30 minutes of ischemia followed by 4 hours of reperfusion. Prior to occlusion, rats were treated with 1 mg/kg P7E4 (a mAb against rat **mannose binding lectin-MBL** A) or GS-1 (an isotype control antibody). Following 4 hours of reperfusion, the hearts were removed, washed in saline and the area at risk was frozen in liquid nitrogen. Total RNA was isolated using the acid-guanidinium thiocyanate extraction procedure and subjected to DNAse treatment. Semi-quantitative RT-PCR was performed to assess mRNA levels of ICAM-1, TNF-alpha, iNOS, eNOS, SOD (Cu/Zn and Mn) GM-CSF and IL-1 alpha, in sham operated animals and those treated with GS-1 or P7E4 as described above. Ischemia-reperfusion resulted in the increased expression of ICAM-1*, iNOS*, TNF-alpha*, GM-CSF* and IL-alpha* compared to sham operated controls. Inhibition of **MBL** A attenuated the increased expression for ICAM-1*, GM-CSF*, IL-1 alpha* and iNOS. These data suggest that inhibition of **MBL** A and the lectin **complement** pathway results in the attenuation of inflammatory gene expression and that this gene regulation may be partially responsible for the protection from MI/R injury, * $p < 0.05$.

L4 ANSWER 8 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:196301 BIOSIS
DOCUMENT NUMBER: PREV200100196301

TITLE: Inhibition of **mannose bin**
lectin reduces myocardial reperfusion injury: A
role for the lectin **complement** pathway in
cardiovascular disease.
AUTHOR(S): Jordan, James E. (1); **Stahl, Gregory L.** (1)
CORPORATE SOURCE: (1) Dept. of Anesthesia, CET and RI, Brigham and Women's
Hospital, Boston, MA USA
SOURCE: Journal of the American College of Cardiology, (February,
2001) Vol. 37, No. 2 Supplement A, pp. 378A. print.
Meeting Info.: 50th Annual Scientific Session of the
American College of Cardiology Orlando, Florida, USA March
18-21, 2001
ISSN: 0735-1097.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L4 ANSWER 9 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:276725 BIOSIS
DOCUMENT NUMBER: PREV200100276725
TITLE: A peptide mimic of N-acetyl-D-glucosamine inhibits the
lectin **complement** pathway following endothelial
oxidative stress.
AUTHOR(S): Montalto, Michael C. (1); **Collard, Charles D.** (1)
; Buras, Jon A.; Reenstra, Wende R.; Geis, David; Rother,
Russell P.; **Stahl, Gregory L.** (1)
CORPORATE SOURCE: (1) Brigham and Women's Hospital, 75 Francis Street,
Boston, MA, 02115 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A339.
print.
Meeting Info.: Annual Meeting of the Federation of American
Societies for Experimental Biology on Experimental Biology
2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Complement** plays a significant role in mediating endothelial
damage following oxidative stress. We have previously demonstrated that
the lectin **complement** pathway (LCP), which is initiated by
mannose binding lectin (**MBL**)
deposition, is largely responsible for activating **complement**
after endothelial oxidative stress. Identifying functional inhibitors of
MBL will be useful in characterizing the role of the LCP following
periods of oxidative stress. To date, peptide analogues specific for
MBL have not been identified. The human cytokeratin peptide,
SFGSGFGGGY, has previously been identified as a molecular mimic of
N-acetyl-D-glucosamine (GlcNAc), a natural ligand of **MBL**. Thus,
we hypothesized that the sequence SFGSGFGGGY would specifically bind
MBL and functionally inhibit the LCP. Using a BIAcore 3000 optical
biosensor, we performed competition experiments to demonstrate that the
peptide SFGSGFGGGY can inhibit binding of recombinant human **MBL**
to GlcNAc in a concentration dependent manner. Solution affinity data
generated by BIAcore indicate this peptide binds to **MBL** with an
affinity (KD) of 5 X 10-5 M. Pretreatment of human serum (30%) with the
GlcNAc-mimicking peptide (10 - 50 mug/ml) significantly attenuated C3
deposition on oxidatively stressed endothelial cells in a dose dependent
manner, as demonstrated by ELISA. Confocal microscopy experiments revealed
that **complement** inhibition coincided with a decrease in
MBL deposition. Additionally, this peptide significantly
attenuated the **complement**-dependent expression of vascular cell
adhesion molecule (VCAM)-1 as shown by ELISA. These data indicate that a
short peptide sequence that mimics GlcNAc can specifically bind to
MBL, with a physiologically relevant affinity, and functionally
inhibit the pro-inflammatory action of the LCP on oxidatively stressed
endothelial cells. Further, this is the first report to demonstrate that
MBL is capable of specifically binding a non-carbohydrate ligand.

L4 ANSWER 10 OF 21 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2001209693 MEDLINE
DOCUMENT NUMBER: 21195380 PubMed ID: 11298833
TITLE: Isolation, cloning and functional characterization of
porcine **mannose-binding lectin**
.
AUTHOR: Agah A; Montalto M C; Young K; **Stahl G L**
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion
Injury, Department of Anesthesiology, Perioperative and
Pain Medicine, Brigham & Women's Hospital, Harvard Medical
School, Boston, MA 02115, USA.
CONTRACT NUMBER: HL52886 (NHLBI)
SOURCE: IMMUNOLOGY, (2001 Mar) 102 (3) 338-43.
Journal code: GH7; 0374672. ISSN: 0019-2805.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010510
AB **Binding** of **mannose-binding lectin**
(**MBL**), a C-type lectin, and its associated serine proteases,
MASP-1 and MASP-2, to cell surface carbohydrates activates the lectin
complement pathway. As **MBL** plays an important role in
innate immunity, it has been cloned and characterized in several species.
While the pig may be used as a source of organs/tissues for
xenotransplantation, little is known about its **MBL**, thus, we
report the isolation of three monomeric forms of **MBL** from
porcine serum. Sodium dodecyl sulphate-polyacrylamide gel electrophoresis
and Coomassie staining of reduced porcine **MBL** revealed the
presence of three monomeric forms with approximate molecular masses of 30
000, 32 000 and 34 000. Protein sequencing identified these monomeric
forms as one single protein, suggesting post-translational modification.
Western blot analysis demonstrated the cross-reactivity of anti-human
MBL polyclonal antibody with porcine **MBL**. A full-length
porcine liver **MBL** cDNA was isolated and the predicted amino acid
sequence exhibited 64.9% identity with human **MBL** and 50.2% and
56.7% identity with rat A and C **MBL**, respectively. Furthermore,
Northern blot analysis demonstrated the presence of a single (
approximately 1.4-1.6 kilobase pair) transcript in porcine liver. Addition
of purified porcine **MBL** to **MBL**-deficient human sera
augmented N-acetylglucosamine inhibitable C3 deposition to mannan-coated
plates in a dose-dependent manner. Taken together, these data demonstrate

that porcine and human **MBL** are highly con d, sharing structural and functional characteristics.

L4 ANSWER 11 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:276723 BIOSIS
DOCUMENT NUMBER: PREV200100276723
TITLE: Isolation and characterization of anti-rat **mannose binding lectin** antibodies.
AUTHOR(S): Jordan, James E. (1); Morrissey, Margaret A. (1); **Stahl, Gregory L. (1)**
CORPORATE SOURCE: (1) Brigham and Women's Hospital, 75 Francis St., Boston, MA, 02115 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A338. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Complement** is a major participant in post-ischemic reperfusion injury. The classical and alternative **complement** pathways have been extensively studied; however, the role of the lectin **complement** pathway (LCP) in inflammation has not been investigated since inhibitors of **MBL** have not been described. In order to delineate the participation of the LCP, we generated antibodies to rat **mannose binding lectin** (**MBL**). In this study, antibodies (Ab) were produced and characterized for potential use in further dissecting the role of the LCP in rat models of human disease. Polyclonal and monoclonal antibodies were produced in rabbits and mice using purified **MBL** isolated from rat sera by affinity chromatography and standard immunization techniques. All antibodies were screened using 2 different ELISA systems (binding to mannan or BSA coupled to N-acetyl-D-glucosamine; BSA-GlcNAc) to detect **complement** activation and deposition of C3. Monoclonal Ab P7E4 inhibited C3 deposition onto BSA-GlcNAc coated plates in a concentration-dependent manner with maximal inhibition (apprx80%) occurring at 10 mug/mL. Similarly, Fab fragments of the polyclonal antibody inhibited **complement** deposition in a concentration dependent manner. Western blot analysis was performed to determine the isoform(s) of rat **MBL** that the antibodies recognized. While the polyclonal Ab recognized multiple oligomers of A and C isoforms under non-reducing conditions, mAb P7E4 only recognized the A isoform. Similar results were obtained under reducing conditions in that the polyclonal Ab recognized both forms while only the A isoform was recognized by P7E4. These data confirm that the antibodies recognize rat **mannose binding lectin**, inhibit the function of **MBL** and may serve as useful reagents in studying the lectin **complement** pathway in rat models of human disease.

L4 ANSWER 12 OF 21 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 2001422305 MEDLINE
DOCUMENT NUMBER: 21167477 PubMed ID: 11266613
TITLE: Ulex europaeus agglutinin II (UEA-II) is a novel, potent inhibitor of **complement** activation.
AUTHOR: Lekowski R; **Collard C D**; Reenstra W R; **Stahl G L**
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: GM-07592 (NIGMS)
HL-03854 (NHLBI)
HL-52886 (NHLBI)
HL-56086 (NHLBI)
SOURCE: PROTEIN SCIENCE, (2001 Feb) 10 (2) 277-84.
Journal code: BNW; 9211750. ISSN: 0961-8368.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010730
Last Updated on STN: 20010730
Entered Medline: 20010726
AB **Complement** is an important mediator of vascular injury following oxidative stress. We recently demonstrated that **complement** activation following endothelial oxidative stress is mediated by **mannose-binding lectin** (**MBL**) and activation of the lectin **complement** pathway. Here, we investigated whether nine plant lectins which have a binding profile similar to that of **MBL** competitively inhibit **MBL** deposition and subsequent **complement** activation following human umbilical vein endothelial cell (HUVEC) oxidative stress. HUVEC oxidative stress (1% O(2), 24 hr) significantly increased Ulex europaeus agglutinin II (UEA-II) binding by 72 +/- 9% compared to normoxic cells. UEA-II inhibited **MBL** binding to HUVEC in a concentration-dependent manner following oxidative stress. Further, **MBL** inhibited UEA-II binding to HUVEC in a concentration-dependent manner following oxidative stress, suggesting a common ligand. UEA-II (< or = 100 micromol/L) did not attenuate the hemolytic activity, nor did it inhibit C3a des Arg formation from alternative or classical **complement** pathway-specific hemolytic assays. C3 deposition (measured by ELISA) following HUVEC oxidative stress was inhibited by UEA-II in a concentration-dependent manner (IC(50) = 10 pmol/L). UEA-II inhibited C3 and **MBL** co-localization (confocal microscopy) in a concentration-dependent manner on HUVEC following oxidative stress (IC(50) approximately 1 pmol/L). Finally, UEA-II significantly inhibited **complement**-dependent neutrophil chemotaxis, but failed to inhibit fMLP-mediated chemotaxis, following endothelial oxidative stress. These data demonstrate that UEA-II is a novel, potent inhibitor of human **MBL** deposition and **complement** activation following human endothelial oxidative stress.

L4 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:420986 CAPLUS
DOCUMENT NUMBER: 133:57580
TITLE: Methods and products for regulating lectin **complement** pathway associated **complement** activation
INVENTOR(S): **Stahl, Gregory L.; Collard, Charles D.**

PATENT ASSIGNEE(S): Brigham and Women's H[redacted]tal, Inc., USA
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000035483	A1	20000622	WO 1999-US29919	19991215
W: CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1140171	A1	20011010	EP 1999-967362	19991215
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-112390P P 19981215
WO 1999-US29919 W 19991215

AB The invention relates to methods and products for regulating lectin **complement** pathway assocd. **complement** activation. The methods include both in vitro and in vivo methods for inhibiting lectin **complement** pathway assocd. **complement** activation. The methods are accomplished by contacting a mammalian cell having surface exposed **MBL** ligand with an effective amt. of a mannan binding lectin inhibitor to inhibit lectin **complement** pathway assocd. **complement** activation. The mannan binding lectin inhibitor may be administered to a subject to prevent cellular injury mediated by lectin **complement** pathway assocd. **complement** activation. The products of the invention include compns. of a mannan binding lectin inhibitor. The mannan binding lectin inhibitor is an isolated mannan binding lectin binding peptide that selectively binds to a human mannan binding lectin epitope and that inhibits lectin **complement** pathway assocd. **complement** activation. The products also include hybridoma cell lines and pharmaceutical compns.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 14 OF 21 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 2000255148 MEDLINE
DOCUMENT NUMBER: 20255148 PubMed ID: 10793066
TITLE: **Complement** activation after oxidative stress: role of the lectin **complement** pathway.
AUTHOR: **Collard C D**; Vakeva A; Morrissey M A; Agah A; Rollins S A; Reenstra W R; Buras J A; Meri S; **Stahl G L**
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: HL-03854 (NHLBI)
HL-52886 (NHLBI)
SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (2000 May) 156 (5) 1549-56.
Journal code: 3RS; 0370502. ISSN: 0002-9440.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000616
Last Updated on STN: 20000616
Entered Medline: 20000602

AB The **complement** system plays an important role in mediating tissue injury after oxidative stress. The role of **mannose-binding lectin** (**MBL**) and the lectin **complement** pathway (LCP) in mediating **complement** activation after endothelial oxidative stress was investigated. iC3b deposition on hypoxic (24 hours; 1% O(2))/reoxygenated (3 hours; 21% O(2)) human endothelial cells was attenuated by N-acetyl-D-glucosamine or D-mannose, but not L-mannose, in a dose-dependent manner. Endothelial iC3b deposition after oxidative stress was also attenuated in **MBL** -deficient serum. Novel, functionally inhibitory, anti-human **MBL** monoclonal antibodies attenuated **MBL**-dependent C3 deposition on mannan-coated plates in a dose-dependent manner. Treatment of human serum with anti-**MBL** monoclonal antibodies inhibited **MBL** and C3 deposition after endothelial oxidative stress. Consistent with our in vitro findings, C3 and **MBL** immunostaining throughout the ischemic area at risk increased during rat myocardial reperfusion in vivo. These data suggest that the LCP mediates **complement** activation after tissue oxidative stress. Inhibition of **MBL** may represent a novel therapeutic strategy for ischemia/reperfusion injury and other **complement**-mediated disease states.

L4 ANSWER 15 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:389415 BIOSIS
DOCUMENT NUMBER: PREV200000389415
TITLE: Endothelial oxidative stress increases cytokeratin 1 (K1) expression and human **mannose-binding lectin** (**MBL**) deposition.
AUTHOR(S): **Collard, C. D. (1)**; Montalto, M. (1); **Stahl, G. L. (1)**
CORPORATE SOURCE: (1) Brigham and Women's Hospital, CET and RI, Harvard Medical School, Boston, MA USA
SOURCE: Immunopharmacology, (August, 2000) Vol. 49, No. 1-2, pp. 85. print.
Meeting Info.: XVIIIth International Complement Workshop Salt Lake City, Utah, USA July 23-27, 2000
ISSN: 0162-3109.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L4 ANSWER 16 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:389411 BIOSIS
DOCUMENT NUMBER: PREV200000389411
TITLE: Characterization of monoclonal antibodies (mAb) against native and recombinant human **mannose-binding lectin** (**MBL**.
AUTHOR(S): Zhao, H. (1); **Stahl, G. L. (1)**
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, Boston, MA USA
SOURCE: Immunopharmacology, (August, 2000) Vol. 49, No. 1-2, pp. 83. print.

Meeting Info.: XVIIIth International Complement Workshop
Salt Lake City, Utah, USA July 23-27, 2000
ISSN: 0162-3109.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L4 ANSWER 17 OF 21 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 2000162174 MEDLINE
DOCUMENT NUMBER: 20162174 PubMed ID: 10698348
TITLE: **Complement** activation following oxidative stress.
AUTHOR: **Collard C D**; Lekowski R; Jordan J E; Agah A; **Stahl G L**
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: GM-07592 (NIGMS)
HL-03854 (NHLBI)
HL-52886 (NHLBI)
+
SOURCE: MOLECULAR IMMUNOLOGY, (1999 Sep-Oct) 36 (13-14) 941-8.
Ref: 70
Journal code: NG1; 7905289. ISSN: 0161-5890.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000327
Last Updated on STN: 20000327
Entered Medline: 20000316
AB It is clear that **complement** plays an important role in the inflammatory process following oxidative stress in cellular and animal models. Clinical trials underway with novel **complement** inhibitors will establish the potential therapeutic benefit of **complement** inhibition in human disease. For as much as we understand about the role of **complement** in disease states, many questions remain. How is **complement** activated on endothelial cells following oxidative stress? What is the ligand for **MBL** on endothelial cells following oxidative stress? Will inhibition of **MBL** provide tissue protection to the extent observed with other **complement** inhibitors such as sCR1 or anti-C5 mAbs? These questions and more will undoubtedly be answered in the next millennium.

L4 ANSWER 18 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:384292 BIOSIS
DOCUMENT NUMBER: PREV199900384292
TITLE: **Mannose-binding lectin** co-localizes with **complement** in atherosclerotic human coronary arteries: A novel role for the lectin **complement** pathway in human cardiovascular disease.
AUTHOR(S): Vakeva, A. (1); **Collard, C. D.**; Laine, P.; Morse, D. S.; Paavonen, T.; Meri, S. (1); Kovanen, P.; **Stahl, G. L.**
CORPORATE SOURCE: (1) Haartman Institute, Department of Bacteriology and Immunology, University of Helsinki, Helsinki Finland
SOURCE: Molecular Immunology, (March-April, 1999) Vol. 36, No. 4-5, pp. 302.
Meeting Info.: 7th European Meeting on Complement in Human Disease Helsinki, Finland June 17-20, 1999
ISSN: 0161-5890.
DOCUMENT TYPE: Conference
LANGUAGE: English

L4 ANSWER 19 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:167550 BIOSIS
DOCUMENT NUMBER: PREV199900167550
TITLE: Purification, characterization and cDNA sequencing of porcine **mannose-binding lectin** (**MBL**.
AUTHOR(S): Agah, A.; Young, K.; **Stahl, G. L.**
CORPORATE SOURCE: CET and RI, Dep. Anesthesia, Brigham and Women's Hosp., Harvard Med. Sch., Boston, MA 02115 USA
SOURCE: FASEB Journal, (March 12, 1999) Vol. 13, No. 4 PART 1, pp. A284.
Meeting Info.: Annual Meeting of the Professional Research Scientists for Experimental Biology 99 Washington, D.C., USA April 17-21, 1999
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

L4 ANSWER 20 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:395748 BIOSIS
DOCUMENT NUMBER: PREV199900395748
TITLE: Endothelial reoxygenation activates the lectin **complement** pathway: Inhibition with anti-human **mannose binding lectin** (**MBL**) therapy.
AUTHOR(S): **Collard, C. D. (1)**; Agah, A. (1); Buras, J. A. (1); Reenstra, W. R. (1); Morrissey, M. M. (1); **Stahl, G. L. (1)**
CORPORATE SOURCE: (1) Center for Experimental Therapeutics and Reperfusion Injury, Dept. of Anesthesiology, Pain and Perioperative Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA USA
SOURCE: Molecular Immunology, (March April, 1999) Vol. 36, No. 4-5, pp. 278.
Meeting Info.: 7th European Meeting on Complement in Human Disease Helsinki, Finland June 17-20, 1999
ISSN: 0161-5890.
DOCUMENT TYPE: Conference
LANGUAGE: English

L4 ANSWER 21 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:24535 BIOSIS
DOCUMENT NUMBER: PREV200000024535
TITLE: Ulex europaeus agglutinin II (UEA-II) is a novel, potent inhibitor of **complement** activation on human endothelial cells.

AUTHOR(S): Lekowski, Robert W. (1); R ra, Wende R.; **Collard, Charles D.; Stahl, Gregory L.**
CORPORATE SOURCE: (1) Brigham and Women's Hosp, Harvard Med Sch, Boston, MA USA
SOURCE: Circulation, (Nov. 2, 1999) Vol. 110, No. 18 SUPPL., pp. I.259.
Meeting Info.: 72nd Scientific Sessions of the American Heart Association Atlanta, Georgia, USA November 7-10, 1999
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

=> s (3f8 or 2a9 or hmbl1.2 or (HB-12621 or HB-12620 or hb-12619) and ((MBL) or (mannose (1N) binding (1N) lectin))
UNMATCHED LEFT PARENTHESIS '(3F8'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s (3f8 or 2a9 or hmbl1.2 or HB-12621 or HB-12620 or hb-12619) and ((MBL) or (mannose (1N) binding (1N) lectin))
L5 3 (3F8 OR 2A9 OR HMBL1.2 OR HB-12621 OR HB-12620 OR HB-12619) AND ((MBL) OR (MANNOSE (1N) BINDING (1N) LECTIN))

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 3 DUP REM L5 (0 DUPLICATES REMOVED)

=> dis l6 1-3 ibib abs

L6 ANSWER 1 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:267818 BIOSIS
DOCUMENT NUMBER: PREV200100267818
TITLE: Epitope mapping monoclonal antibodies against human **mannose binding lectin.**
AUTHOR(S): Zhao, Hui (1); Stahl, Gregory L. (1)
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA, 02115 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A685. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB **MBL** plays an important role in complement activation following endothelial oxidative stress. We have generated a panel of monoclonal antibodies (**3F8**, **hMBL1.2** and **2A9**) against human **MBL**. These antibodies are functional inhibitors, which can attenuate **MBL**-dependent C3 deposition after endothelial oxidative stress. The affinity of Fab fragments and whole IgG antibodies to **MBL** were very similar. However, Fab fragments of **hMBL1.2** or **2A9** did not inhibit C3 deposition in a **MBL** dependent assay. Further, F(ab)2 fragments of **2A9** or **hMBL1.2** were functionally much less effective compared to whole IgG, suggesting that steric hindrance of these two antibodies are important for their inhibition of **MBL** binding. Fab and F(ab)2 fragments of **3F8**, on the other hand, were functionally as effective as the whole IgG. All three functional antibodies (**3F8**, **hMBL1.2** and **2A9**) bind to the carbohydrate recognition domain (CRD) of **MBL** based on protein sequencing and Western analysis of proteolytic fragments of **MBL**. **MBL** constructs consisting of sequential deletion of N- or C-terminal amino acids of the CRD region showed that the antibodies recognized different epitopes. Two disulfide bonds within the **MBL** monomer. Cys155 to Cys244 and Cys222 to Cys236 aid in stabilization of the CRD. Single, double and triple mutations of these cystidines showed that the disulfide bonds played a role in forming discontinuous epitopes for **3F8** and **hMBL1.2**. Epitope maps of these antibodies were further confirmed by biopanning using the FliTrx random peptide display library. Generating monoclonal antibodies against **MBL** will aid in the structure/function analysis of **MBL** and its role in inflammatory diseases.

L6 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:420986 CAPLUS
DOCUMENT NUMBER: 133:57580
TITLE: Methods and products for regulating lectin complement pathway associated complement activation
INVENTOR(S): Stahl, Gregory L.; Collard, Charles D.
PATENT ASSIGNEE(S): Brigham and Women's Hospital, Inc., USA
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000035483	A1	20000622	WO 1999-US29919	19991215
W: CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1140171	A1	20011010	EP 1999-967362	19991215
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-112390P P 19981215
WO 1999-US29919 W 19991215

AB The invention relates to methods and products for regulating lectin complement pathway assocd. complement activation. The methods include both in vitro and in vivo methods for inhibiting lectin complement pathway assocd. complement activation. The methods are accomplished by contacting a mammalian cell having surface exposed **MBL** ligand with an effective amt. of a mannan binding lectin inhibitor to inhibit lectin complement pathway assocd. complement activation. The mannan binding lectin inhibitor may be administered to a subject to prevent cellular injury mediated by lectin complement pathway assocd. complement activation. The products of the invention include compns. of a mannan binding lectin inhibitor. The mannan binding lectin inhibitor is an isolated mannan binding lectin binding peptide that selectively binds to a human mannan binding lectin epitope and that inhibits lectin complement pathway assocd. complement activation. The products also include

hybridoma cell lines and pharmaceutical c[redacted]s.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:389411 BIOSIS
DOCUMENT NUMBER: PREV200000389411
TITLE: Characterization of monoclonal antibodies (mAb) against native and recombinant human **mannose-binding lectin** (**MBL**.
AUTHOR(S): Zhao, H. (1); Stahl, G. L. (1)
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, Boston, MA USA
SOURCE: Immunopharmacology, (August, 2000) Vol. 49, No. 1-2, pp. 83. print.
Meeting Info.: XVIIIth International Complement Workshop Salt Lake City, Utah, USA July 23-27, 2000
ISSN: 0162-3109.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
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